L22 ANSWER 1 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 1

AN 1999:297320 BIOSIS

DN PREV199900297320

TI The antiangiogenic agents TNP-470 and 2-methoxyestradiol inhibit the growth of **angiosarcoma** in mice.

AU Arbiser, Jack L. [Reprint author]; Panigrathy, Dipak; Klauber, Nancy; Rupnick, Maria; Flynn, Evelyn; Udagawa, Taturo; D'Amato, Robert J.

CS Department of Dermatology, Emory University School of Medicine, WMB 5309, Atlanta, GA, 30322, USA

SO Journal of the American Academy of Dermatology, (**June, 1999**)
Vol. 40, No. 6 PART 1, pp. 925-929. print.
ISSN: 0190-9622.

DT Article

LA English

ED Entered STN: 5 Aug 1999
Last Updated on STN: 5 Aug 1999

AB Background: Endothelial malignancies, such as **angiosarcoma** and hemangioendothelioma, are often resistant to chemotherapy and surgery, and may result in death. Improved means of therapy are needed for these disorders. Objective: We wanted to determine whether **angiosarcoma** can be treated with **angiogenesis** inhibitors in mice. Methods: Mice were inoculated with a cell line that gives rise to **angiosarcoma** and were treated with the **angiogenesis** inhibitors 2-methoxyestradiol and TNP-470. Response to therapy was monitored by measurement of tumors. Results: TNP-470 caused an 84% reduction in tumor size, and 2-methoxyestradiol caused a 68% reduction in tumor size. Conclusion: **Angiogenesis** inhibitors are highly effective in treatment of **angiosarcoma** in mice. Clinical trials of these agents in humans with **angiosarcoma** and hemangioendothelioma are warranted.

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      (FILE 'HOME' ENTERED AT 07:30:40 ON 31 OCT 2003)

      FILE 'ADISCTI, ADISINSIGHT, ADISNEWS, BIOSIS, BIOTECHNO, CANCERLIT,
      CAPLUS, CEN, DISSABS, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL,
      DRUGU, EMBAL, EMBASE, ESBIOBASE, IFIPAT, IPA, JICST-EPLUS, KOSMET,
      LIFESCI, MEDICONF, MEDLINE, NAPRALERT, NLDB, NUTRACEUT, ..' ENTERED AT
      07:30:52 ON 31 OCT 2003
L1         86627 S (BASIC AND FIBROBLAST AND GROWTH AND FACTOR)
L2            10 S L1 AND DEMETHOXYCURCUMIN
L3             6 DUP REM L2 (4 DUPLICATES REMOVED)
L4             2 S L3 AND PD<2000
L5          1358 S VENOUS (W) ULCER
L6           484 S L5 AND PD<2000
L7            10 S L6 AND ANGIOGENESIS
L8            10 DUP REM L7 (0 DUPLICATES REMOVED)
L9             0 S L8 AND (CURCUMIN OR CURCUMINOID OR DEMETHOXYCURCUMIN)
L10            1 S L7 AND L1
L11            1 S (BASIC (W) FIBROBLAST (W) GROWTH (W) FACTOR) AND L8
L12           51 S L5 (P) ANGIOGENESIS
L13           46 DUP REM L12 (5 DUPLICATES REMOVED)
L14            2 S L13 AND PD<2000
L15          229 S ANGIOSARCOMA (P) ANGIOGENESIS
L16           59 S L15 AND PD<2000
L17            2 S (BASIC (W) FIBROBLAST (W) GROWTH (W) FACTOR) AND L16
L18           57 S L16 NOT L17
L19            0 S L18 AND (CURCUMINOID OR DEMETHOXYCURCUMIN OR CURCUMIN)
L20           38 S L18 AND (ANGIOGENESIS OR ANGIOSARCOMA)/TI
L21           19 S L20 AND (ANGIOGENESIS OR ANGIOSARCOMA)/AB
L22           12 DUP REM L21 (7 DUPLICATES REMOVED)
L23           12 S ANGIOSARCOMA AND (CURCUMINOID OR DEMETHOXYCURCUMIN OR CURCUMI
L24            8 DUP REM L23 (4 DUPLICATES REMOVED)
L25            0 S L24 AND PD<2000

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L4 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:232405 BIOSIS
DN PREV199800232405
TI Inhibition of angiogenic differentiation of human umbilical vein endothelial cells by **curcumin**.
AU Thaloor, Deepa; Singh, Anoop K.; Sidhu, Gurmel S.; Prasad, Paruchuri V.; Kleinman, Hynda K.; Maheshwari, Radha K. [Reprint author]
CS Cent. Combat Casualty Life Sustainment Res., Dep. Pathol., Uniformed Serv. Univ. Health Sci., Bethesda, MD 20814, USA
SO Cell Growth and Differentiation, (**April, 1998**) Vol. 9, No. 4, pp. 305-312. print.
ISSN: 1044-9523.
DT Article
LA English
ED Entered STN: 20 May 1998
Last Updated on STN: 20 May 1998
TI Inhibition of angiogenic differentiation of human umbilical vein endothelial cells by **curcumin**.
SO Cell Growth and Differentiation, (**April, 1998**) Vol. 9, No. 4, pp. 305-312. print.
ISSN: 1044-9523.
AB Angiogenesis is a crucial step in the growth and metastasis of cancers. **Curcumin** inhibits tumor initiation and growth. We analyzed the effect of **curcumin** on endothelial cell migration, attachment, and tube formation on Matrigel. **Curcumin** had no effect on endothelial cell migration or attachment to either plastic or Matrigel. **Curcumin** treatment resulted in a dose-dependent inhibition of tube formation when the cells were treated before plating or at the time of plating on Matrigel. **Curcumin** treatment also caused the preformed tubes to break down. **Curcumin** inhibited angiogenesis in a s.c. Matrigel plug model in mice. The role of metalloproteinases has been shown to be important in angiogenesis; therefore, zymography was performed to determine whether **curcumin** affected protease activity. Zymographs of **curcumin**-treated culture supernatants showed a decrease in the gelatinolytic activities of secreted 53- and 72-kDa metalloproteinases. Western and Northern analysis showed a dose-dependent decrease in the protein expression and transcript of 72 kDa, indicating that **curcumin** may be exerting its inhibitory effect at both the transcriptional and posttranscriptional level. These findings suggest that **curcumin** acts as an angiogenesis inhibitor by modulating protease activity during endothelial morphogenesis. **Curcumin** could be developed as an **antiangiogenic** drug.
IT Major Concepts
Cardiovascular System (Transport and Circulation); Pharmacology
IT Chemicals & Biochemicals
curcumin: cardiovascular-drug, **antiangiogenic** agent, potential anticancer agent
RN 458-37-7 (**curcumin**)

=>

L8 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:243121 BIOSIS
DN PREV199800243121
TI Evidence for angiostatic activity of **curcumin**.
AU Mohan, R.; Ashton, P.; Kasahara, N.; Pham, B. Q.; Russo, L. A.; Fini, M. E.
CS Vision Res. Lab., New England Med. Center/Tufts Univ. Sch. Med., Boston, MA, USA
SO IOVS, (**March 15, 1998**) Vol. 39, No. 4, pp. S895. print.
Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology. Fort Lauderdale, Florida, USA. May 10-15, 1998. Association for Research in Vision and Ophthalmology.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 4 Jun 1998
Last Updated on STN: 4 Jun 1998
CC Sense organs - General and methods 20001
Cytology - General 02502
Biochemistry studies - General 10060
Cardiovascular system - General and methods 14501
Pharmacology - General 22002
General biology - Symposia, transactions and proceedings 00520
IT Major Concepts
Cardiovascular System (Transport and Circulation); Pharmacology; Sense Organs (Sensory Reception)
IT Parts, Structures, & Systems of Organisms
corneal fibroblast cells: sensory system, culture
IT Chemicals & Biochemicals
basic fibroblast growth factor;
curcumin: AP-1 inhibitor, angiostatic activity; AP-1: transcription factor; NF-kappa B [nuclear factor-kappa B]: transcription factor; PMA [phorbol 12-myristate 13-acetate]
IT Miscellaneous Descriptors
Meeting Abstract
ORGN Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rabbit
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
RN 106096-93-9 (**basic fibroblast growth factor**)
458-37-7 (**curcumin**)
62-38-4Q (PMA)
64-13-1Q (PMA)
16561-29-8Q (PMA)
25087-26-7Q (PMA)
78565-16-9Q (PMA)
276704-22-4Q (PMA)
62-38-4Q (phorbol 12-myristate 13-acetate)
64-13-1Q (phorbol 12-myristate 13-acetate)
16561-29-8Q (phorbol 12-myristate 13-acetate)
25087-26-7Q (phorbol 12-myristate 13-acetate)
78565-16-9Q (phorbol 12-myristate 13-acetate)
276704-22-4Q (phorbol 12-myristate 13-acetate)

=>

L6 ANSWER 1 OF 13 USPATFULL on STN
AN 2003:283096 USPATFULL
TI Composition for the treatment of damaged tissue
IN Dack, Kevin Neil, Kent, UNITED KINGDOM
Davies, Michael John, Kent, UNITED KINGDOM
Fish, Paul Vincent, Kent, UNITED KINGDOM
Huggins, Jonathan Paul, Kent, UNITED KINGDOM
McIntosh, Fraser Stuart, Kent, UNITED KINGDOM
Occleston, Nicholas Laurence, Kent, UNITED KINGDOM
PA Pfizer Inc. (non-U.S. corporation)
PI US 2003199440 A1 20031023
AI US 2002-131985 A1 20020425 (10)
RLI Continuation of Ser. No. US 2000-725295, filed on 29 Nov 2000, PENDING
PRAI GB 1999-30768 19991229
US 2000-186426P 20000302 (60)
DT Utility
FS APPLICATION
LREP PFIZER INC, 150 EAST 42ND STREET, 5TH FLOOR - STOP 49, NEW YORK, NY, 10017-5612
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 19445
TI Composition for the treatment of damaged tissue
AB A pharmaceutical for use in damaged tissue, such as wound, treatment (e.g. healing) is described. The pharmaceutical comprising a composition which comprises: (a) a growth factor; and (b) an inhibitor agent; and optionally (c) a pharmaceutically acceptable carrier, diluent or excipient; wherein the inhibitor agent can inhibit the action of at least one specific adverse protein (e.g. a specific protease) that is upregulated in a damaged tissue, such as a wound, environment.

L6 ANSWER 2 OF 13 USPATFULL on STN
AN 2003:265851 USPATFULL
TI Anti-**angiogenic** peptides
IN Rosenbaum, Jan Susan, Cincinnati, OH, UNITED STATES
Jones, David R., Milford, OH, UNITED STATES
Whitaker, George Brian, West Chester, OH, UNITED STATES
PI US 2003186868 A1 20031002
AI US 2002-263162 A1 20021002 (10)
PRAI US 2001-326712P 20011003 (60)
DT Utility
FS APPLICATION
LREP REGENERON PHARMACEUTICALS, INC, 777 OLD SAW MILL RIVER ROAD, TARRYTOWN, NY, 10591
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 2508
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Anti-**angiogenic** peptides
AB Peptides that specifically interfere with the ability of VEGF.sub.165 to interact with the NP-1 receptor or with a VEGFR-2/NP-1 co-receptor complex are disclosed. The inventive peptides are useful to control pathological **angiogenesis**, such as occurs in cancer and other diseases. The peptides are based on a combination of basic residues contained within Exon 6 of human placental growth factor (PIGF), coupled at the carboxyl terminus to either Exon 8 of VEGF.sub.165 or Exon 7 of PIGF. The peptides behave as antagonists of VEGF.sub.165 signaling through a mechanism that involves competition for VEGF.sub.165 binding at either the VEGFR-2/NP-1 complex or NP-1, without affecting VEGF signaling through other pathways. This binding is sufficient to attenuate pathological **angiogenesis** such as occurs in tumor

growth.

L6 ANSWER 3 OF 13 IFIPAT COPYRIGHT 2003 IFI on STN DUPLICATE 1
AN 10063443 IFIPAT;IFIUDB;IFICDB
TI CURCUMIN AND CURCUMINOID INHIBITION OF **ANGIOGENESIS**; SKIN DISORDERS
INF ARBISER; JACK L., ATLANTA, GA, US
IN ARBISER JACK L
PAF Unassigned
PA Unassigned Or Assigned To Individual (68000)
AG PATREA L PABST ARNALL GOLDEN & GREGORY LLP, 2800 ONE ATLANTIC CENTER, 1201 WEST PEACHTREE STREET, ATLANTA, GA, 303093450
PI US 2002006966 A1 20020117
AI US 1999-345712 19990630
FI US 2002006966 20020117
DT Utility; Patent Application - First Publication
FS CHEMICAL
APPLICATION
GOVI (0001) The United States government has rights in this invention by virtue of grant R03 AR44947 from the National Institutes of Health.
CLMN 16
GI 3 Figure(s).
FIGS. 1A-C describe the effect of curcumin on endothelial cell proliferation in the absence of basic fibroblast growth factor (bFGF; FIG. 1A), in the presence of bFGF (FIG. 1B) and in the absence of bFGF, where the endothelial cells have been transformed (FIG. 1C). The figures are graphs of cell number versus concentration of curcumin (mu M).
FIGS. 2A-2B describe the effect of curcumin on the extent of bFGF-stimulated neovascularization in the mouse cornea (FIG. 2A) , in relation to bFGF-stimulated neovascularization in the absence of curcumin (FIG. 2B). The figures are graphs of vessel length (mm) and sector size (clock hours) comparing curcumin (10 mu M) with control TPCPD, with both in the presence of 80 ng bFGF.
FIGS. 3A and 3B describe the effect of curcumin and other curcuminoids, tetrahydrocurcumin, bisdemethoxycurcumin, and demethoxycurcumin, on corneal neovascularization, as measured by vessel length (FIG. 3A) and by sector size (FIG. 3B).
TI CURCUMIN AND CURCUMINOID INHIBITION OF **ANGIOGENESIS**; SKIN DISORDERS
AB Methods for treating diseases or disorders of the skin which are characterized by **angiogenesis** have been developed using curcumin and curcumin analogs. Based on the results obtained with curcumin, it has been determined that other **angiogenesis** inhibitors can also be used to treat these skin disorders. It has further been discovered that curcumin acts to inhibit **angiogenesis** in part by inhibition of basic fibroblast growth factor (bFGF), and thereby provides a means for treating other disorders characterized by elevated levels of bFGF, such as bladder cancer, using curcumin and other analogues which also inhibit bFGF. Representative skin disorders to be treated include the malignant diseases **angiosarcoma**, hemangioendothelioma, basal cell carcinoma, squamous cell carcinoma, malignant melanoma and Karposi's sarcoma, and the non-malignant diseases or conditions including psoriasis, lymphangiogenesis, hemangioma of childhood, Sturge-Weber syndrome, verruca vulgaris, neurofibromatosis, tuberous sclerosis, pyogenic granulomas, **recessive dystrophic epidermolysis bullosa**, venous ulcers, acne, rosacea, eczema, molluscum contagious, seborrheic keratosis, and actinic keratosis.

L6 ANSWER 4 OF 13 USPATFULL on STN
AN 2002:294281 USPATFULL
TI Medical use of matrix metalloproteinase inhibitors for inhibiting tissue contraction
IN Khaw, Peng Tee, London, UNITED KINGDOM

Schultz, Gregory S., Gainesville, FL, UNITED STATES
PI US 2002164319 A1 20021107
AI US 2002-135934 A1 20020429 (10)
RLI Division of Ser. No. US 1999-368307, filed on 3 Aug 1999, GRANTED, Pat. No. US 6379667 Division of Ser. No. US 1996-716155, filed on 19 Nov 1996, GRANTED, Pat. No. US 6093398 A 371 of International Ser. No. WO 1995-GB576, filed on 16 Mar 1995, UNKNOWN
PRAI GB 1994-5076 19940316
DT Utility
FS APPLICATION
LREP GREENLEE WINNER AND SULLIVAN P C, 5370 MANHATTAN CIRCLE, SUITE 201, BOULDER, CO, 80303
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 1415
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Medical use of matrix metalloproteinase inhibitors for inhibiting tissue contraction
AB The use of an MMP inhibitor, especially a collagenase inhibitor, in the manufacture of a medicament for the treatment of a natural or artificial tissue comprising extracellular matrix components to inhibit contraction of the tissue and methods for the treatment of tissue comprising extracellular matrix components to inhibit contraction.

L6 ANSWER 5 OF 13 USPATFULL on STN
AN 2002:95355 USPATFULL
TI Medical use of matrix metalloproteinase inhibitors for inhibiting tissue contraction
IN Khaw, Peng Tee, London, UNITED KINGDOM
Schultz, Gregory S., Gainesville, FL, United States
PA University of Florida Research Foundation, Gainesville, FL, United States (U.S. corporation)
Moorfields Eye Hospital National Health Service Trust, London, UNITED KINGDOM (non-U.S. corporation)
PI US 6379667 B1 20020430
AI US 1999-368307 19990803 (9)
RLI Division of Ser. No. US 1996-716155, filed on 19 Nov 1996, now patented, Pat. No. US 6093398
PRAI GB 1994-5076 19940316
DT Utility
FS GRANTED
EXNAM Primary Examiner: Nashed, Nashaat T.
LREP Greenlee, Winner and Sullivan, P.C.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 24 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 1374
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Medical use of matrix metalloproteinase inhibitors for inhibiting tissue contraction
AB The use of an MMP inhibitor, especially a collagenase inhibitor, in the manufacture of a medicament for the treatment of a natural or artificial tissue comprising extracellular matrix components to inhibit contraction of the tissue and methods for the treatment of tissue comprising extracellular matrix components to inhibit contraction.

L6 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2003 ACS on STN DUPLICATE 2
AN 2001:12261 CAPLUS
DN 134:66151
TI Curcumin and curcuminoid inhibition of **angiogenesis**
IN Arbiser, Jack L.
PA Emory University, USA
SO PCT Int. Appl., 30 pp.

CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001000201	A1	20010104	WO 2000-US17608	20000627
	W: AU, CA, JP				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 2002006966	A1	20020117	US 1999-345712	19990630
	EP 1196158	A1	20020417	EP 2000-941736	20000627
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	US 2001025034	A1	20010927	US 2001-765491	20010118
PRAI	US 1999-345712	A	19990630		
	WO 2000-US17608	W	20000627		

TI Curcumin and curcuminoid inhibition of **angiogenesis**
AB Methods for treating diseases or disorders of the skin which are characterized by **angiogenesis** have been developed using curcumin and curcumin analogs. Based on the results obtained with curcumin, it has been detd. that other **angiogenesis** inhibitors can also be used to treat these skin disorders. It has further been discovered that curcumin acts to inhibit **angiogenesis** in part by inhibition of basic fibroblast growth factor (bFGF), and thereby provides a means for treating other disorders characterized by elevated levels of bFGF, such as bladder cancer, using curcumin and other analogs which also inhibit bFGF. Representative skin disorders to be treated include the malignant diseases **angiosarcoma**, hemangioendothelioma, basal cell carcinoma, squamous cell carcinoma, malignant melanoma and Kaposi's sarcoma, and the non-malignant diseases or conditions including psoriasis, lymphangiogenesis, hemangioma of childhood, Sturge-Weber syndrome, verruca vulgaris, neurofibromatosis, tuberous sclerosis, pyogenic granulomas, **recessive dystrophic epidermolysis bullosa**, venous ulcers, acne, rosacea, eczema, molluscum contagious, seborrheic keratosis, and actinic keratosis.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 13 IFIPAT COPYRIGHT 2003 IFI on STN DUPLICATE 3
AN 10025024 IFIPAT;IFIUDB;IFICDB
TI CURCUMIN AND CURCUMINOID INHIBITION OF **ANGIOGENESIS**; ADMINISTERING TO THE INDIVISUAL SUFFERING FROM DISORDERS CHARACTERIZED BY ELEVATED LEVELS OF BASIC FIBROBLAST GROWTH FACTOR A CURCUMINOID TO INHIBIT THE BASIC FIBROBLAST GROWTH FACTOR
INF Arbiser; Jack L., Atlanta, GA, US
IN Arbiser Jack L
PAF Emory University
PA Emory University (12419)
AG PATREA L. PABST HOLLAND & KNIGHT LLP, SUITE 2000, ONE ATLANTIC CENTER, 1201 WEST PEACHTREE STREET, N.E., ATLANTA, GA, 30309-3400, US
PI US 2001025034 A1 20010927
AI US 2001-765491 20010118
RLI US 1999-345712 19990630 CONTINUATION
FI US 2001025034 20010927
DT Utility; Patent Application - First Publication
FS CHEMICAL
APPLICATION
GOVI (0001) The United States government has rights in this invention by virtue of grant R03 AR44947 from the National Institutes of Health.
CLMN 16
GI 3 Figure(s).
FIGS. 1A-C describe the effect of curcumin on endothelial cell proliferation in the absence of basic fibroblast growth factor (bFGF;

FIG. 1A), in the presence of bFGF (FIG. 1B) and in the absence of bFGF, where the endothelial cells have been transformed (FIG. 1C). The figures are graphs of cell number versus concentration of curcumin (mu M).

FIGS. 2A-2B describe the effect of curcumin on the extent of bFGF-stimulated neovascularization in the mouse cornea (FIG. 2A) , in relation to bFGF-stimulated neovascularization in the absence of curcumin (FIG. 2B). The figures are graphs of vessel length (mm) and sector size (clock hours) comparing curcumin (10 mu M) with control TPCPD, with both in the presence of 80 ng bFGF.

FIGS. 3A and 3B describe the effect of curcumin and other curcuminoids, tetrahydrocurcumin, bisdemethoxycurcumin, and demethoxycurcumin, on corneal neovascularization, as measured by vessel length (FIG. 3A) and by sector size (FIG. 3B).

TI CURCUMIN AND CURCUMINOID INHIBITION OF **ANGIOGENESIS**; ADMINISTERING TO THE INDIVISUAL SUFFERING FROM DISORDERS CHARACTERIZED BY ELEVATED LEVELS OF BASIC FIBROBLAST GROWTH FACTOR A CURCUMINOID TO INHIBIT THE BASIC FIBROBLAST GROWTH FACTOR

AB Methods for treating diseases or disorders of the skin which are characterized by **angiogenesis** have been developed using curcumin and curcumin analogs. Based on the results obtained with curcumin, it has been determined that other **angiogenesis** inhibitors can also be used to treat these skin disorders. It has further been discovered that curcumin acts to inhibit **angiogenesis** in part by inhibition of basic fibroblast growth factor (bFGF), and thereby provides a means for treating other disorders characterized by elevated levels of bFGF, such as bladder cancer, using curcumin and other analogues which also inhibit bFGF. Representative skin disorders to be treated include the malignant diseases **angiosarcoma**, hemangioendothelioma, basal cell carcinoma, squamous cell carcinoma, malignant melanoma and Karposi's sarcoma, and the non-malignant diseases or conditions including psoriasis, lymphangiogenesis, hemangioma of childhood, Sturge-Weber syndrome, verruca vulgaris, neurofibromatosis, tuberous sclerosis, pyogenic granulomas, **recessive dystrophic epidermolysis bullosa**, venous ulcers, acne, rosacea, eczema, molluscum contagious, seborrheic keratosis, and actinic keratosis.

L6 ANSWER 8 OF 13 USPATFULL on STN
AN 2001:221068 USPATFULL
TI Use of adenosine A3 receptor antagonists to inhibit tumor growth
IN Leung, Edward, Cary, NC, United States
Baraldi, Pier Giovanni, Ferrara, Italy
Borea, Pier Andrea, Ferrara, Italy
Chen, Shih-Fong, Apex, NC, United States
PA King Pharmaceuticals Reseach and Development, Inc., Cary, NC, United States (U.S. corporation)
PI US 6326390 B1 20011204
AI US 1999-377271 19990819 (9)
PRAI US 1998-97852P 19980825 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Jones, Dwayne C.
LREP Roberts Abokhair & Mardula, LLC
CLMN Number of Claims: 39
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 946
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Use of adenosine A3 receptor antagonists to inhibit tumor growth
AB Tumor growth and metastasis can be inhibited by administration of adenosine A.sub.1 and/or A.sub.3 antagonists, preferably A.sub.3 antagonists, to a patient. The antagonists can be, and preferably are, administered in combination with other anti-tumor agents, such as anti-**angiogenic** agents (including adenosine A.sub.2a antagonists)

and/or cytotoxic agents. Because the cytotoxic agents attack the tumor cells themselves, and the anti-**angiogenic** agents prevent the growth of vasculature which would otherwise support the growth of the tumor cells.

L6 ANSWER 9 OF 13 USPATFULL on STN
AN 2000:106073 USPATFULL
TI Enzymatic nucleic acids that cleave C-fos
IN Jarvis, Thale, Boulder, CO, United States
McSwiggen, James A., Boulder, CO, United States
Stinchcomb, Dan T., Ft. Collins, CO, United States
PA Ribozyme Pharmaceuticals, Inc., Boulder, CO, United States (U.S. corporation)
PI US 6103890 20000815
AI US 1997-998099 19971224 (8)
RLI Continuation-in-part of Ser. No. US 1995-373124, filed on 13 Jan 1995, now patented, Pat. No. US 5646042 which is a continuation-in-part of Ser. No. US 1994-245466, filed on 18 May 1994, now abandoned
PRAI US 1997-37658P 19970123 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Brusca, John S.; Assistant Examiner: Shibuya, Mark L.
LREP Lyon & Lyon LLP
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 3659
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Enzymatic nucleic acids that cleave C-fos
AB Enzymatic nucleic acid molecules which cleave c-fos RNA.

L6 ANSWER 10 OF 13 USPATFULL on STN
AN 2000:94697 USPATFULL
TI Medical use of matrix metalloproteinase inhibitors for inhibiting tissue contraction
IN Khaw, Peng Tee, London, United Kingdom
Schultz, Gregory S., Gainesville, FL, United States
PA University of Florida Research Found, Gainesville, FL, United States (U.S. corporation)
Institute of Ophthalmology, London, United Kingdom (non-U.S. corporation)
Moorfields Eye Hospital NHS Trust, London, United Kingdom (non-U.S. corporation)
PI US 6093398 20000725
WO 9524921 19950921
AI US 1996-716155 19961119 (8)
WO 1995-GB576 19950316
19961119 PCT 371 date
19961119 PCT 102(e) date
PRAI GB 1994-5076 19940316
DT Utility
FS Granted
EXNAM Primary Examiner: Achutamurthy, Ponnathapura; Assistant Examiner: Nashed, Nashaat T.
LREP Greenlee, Winner and Sullivan, P.C.
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 24 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 1437
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Medical use of matrix metalloproteinase inhibitors for inhibiting tissue contraction
AB The use of an MMP inhibitor, especially a collagenase inhibitor, in the manufacture of a medicament for the treatment of a natural or artificial

tissue comprising extracellular matrix components to inhibit contraction of the tissue and methods for the treatment of tissue comprising extracellular matrix components to inhibit contraction.

L6 ANSWER 11 OF 13 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 4
AN 1998:257813 BIOSIS
DN PREV199800257813
TI Basic fibroblast growth factor: A missing link between collagen VII, increased collagenase, and squamous cell carcinoma in **recessive dystrophic epidermolysis bullosa**.
AU Arbiser, Jack L. [Reprint author]; Fine, Jo-David; Murrell, Dedee; Paller, Amy; Connors, Susan; Keough, Karen; Marsh, Elizabeth; Folkman, Judah
CS Dep. Dermatol., Harvard Med. Sch., Boston, MA, USA
SO Molecular Medicine (New York), (March, 1998) Vol. 4, No. 3, pp. 191-195. print.
ISSN: 1076-1551.
DT Article
LA English
ED Entered STN: 9 Jun 1998
Last Updated on STN: 12 Aug 1998
TI Basic fibroblast growth factor: A missing link between collagen VII, increased collagenase, and squamous cell carcinoma in **recessive dystrophic epidermolysis bullosa**.
AB Background: Patients with **recessive dystrophic epidermolysis bullosa** (RDEB) have deficiencies of collagen type VII and have elevated levels of fibroblast collagenase, and a greatly increased risk of cutaneous squamous cell carcinoma. Patients with other genetic blistering disorders do not have elevated collagenase or an increased risk of squamous cell carcinoma, despite chronic wounding. The connection between collagen type VII deficiency, increased collagenase, and squamous cell carcinoma is not understood. Materials and Methods: Urine from 81 patients with RDEB (39 patients), junctional epidermolysis bullosa (JEB; 12 patients), and epidermolysis bullosa simplex (EBS; 30 patients), as well as unaffected family members of RDEB patients (33 patients), was tested for the presence of basic fibroblast growth factor (bFGF) using a sensitive radioimmunoassay. These patients included many who were enrolled in the Epidermolysis Bullosa Registry and others who were referred by their physicians. Results: Fifty-one percent of patients with RDEB had elevated levels (>5000 pg/g) of urinary bFGF. In contrast, none of the patients with JEB had elevated levels of bFGF. Twenty-one percent of clinically unaffected family members had elevated levels of bFGF, and 13% of patients with EBS had elevated levels of bFGF. The frequency of elevated bFGF values among all groups was statistically significant ($p = 0.002$), and the levels of bFGF in RDEB patients were significantly elevated compared with those of other groups ($p < 0.05$). Conclusions: We have found that patients with RDEB have elevated levels of bFGF, which may contribute to increased fibroblast collagenase and the development of squamous cell carcinoma. These results suggest a novel treatment for RDEB, namely, **angiogenesis** inhibitors, which may antagonize the effects of bFGF in this disorder. There are currently no other means of treatment for this disorder, which has a high morbidity and mortality rate.

L6 ANSWER 12 OF 13 CANCERLIT on STN DUPLICATE 5
AN 91357222 CANCERLIT
DN 91357222 PubMed ID: 1884860
TI Metastatic squamous cell carcinoma resembling **angiosarcoma** complicating dystrophic epidermolysis bullosa.
AU McGrath J A; Schofield O M; Mayou B J; McKee P H; Eady R A
CS Institute of Dermatology, United Medical School, St. Thomas' Hospital, London, UK.
SO DERMATOLOGICA, (1991) 182 (4) 235-8.
Journal code: 0211607. ISSN: 0011-9075.

CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS MEDLINE; Priority Journals
OS MEDLINE 91357222
EM 199110
ED Entered STN: 19941107
Last Updated on STN: 19941107
TI Metastatic squamous cell carcinoma resembling **angiosarcoma** complicating dystrophic epidermolysis bullosa.
AB We report a patient with generalized **recessive dystrophic epidermolysis bullosa** (RDEB) who developed 3 squamous cell carcinomas. The tumours appeared simultaneously at acral sites on both upper limbs and were poorly differentiated. Despite surgery and radiotherapy the patient died from metastatic disease within 6 months of presentation. This case highlights many of the typical features of this complication of RDEB, including the overall poor prognosis. Of particular interest was the histology of one of the tumours which caused diagnostic difficulties: haematoxylin and eosin staining suggested an **angiosarcomatous** pathology, but the use of immunocytochemistry proved that the tumour was a squamous cell carcinoma in origin.

L6 ANSWER 13 OF 13 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1983:238423 BIOSIS
DN PREV198375088423; BA75:88423
TI COLCHICINE INDUCED MODULATION OF COLLAGENASE IN HUMAN SKIN FIBROBLAST CULTURES 2. A PROBE FOR DEFECTIVE REGULATION IN EPIDERMOLYSIS BULLOSA.
AU BAUER E A [Reprint author]; VALLE K-J; ESTERLY N B
CS DIVISION OF DERMATOLOGY, WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, CAMPUS BOX 8123, 4950 AUDUBON BOULEVARD, ST LOUIS, MISSOURI 63110, USA
SO Journal of Investigative Dermatology, (1982) Vol. 79, No. 6, pp. 403-407. CODEN: JIDEAE. ISSN: 0022-202X.
DT Article
FS BA
LA ENGLISH
TI COLCHICINE INDUCED MODULATION OF COLLAGENASE IN HUMAN SKIN FIBROBLAST CULTURES 2. A PROBE FOR DEFECTIVE REGULATION IN EPIDERMOLYSIS BULLOSA.
AB The addition of colchicine to cultures of normal human skin fibroblasts produces a significant stimulation of collagenase. Because this finding implies a role for the microtubule system in the regulation of normal collagenase synthesis, colchicine was used as a probe for aberrations in this enzymne in epidermolysis bullosa. In fibroblast cultures from the dominant simplex, dominant dystrophic and recessive letalis forms of epidermolysis bullosa, 10-6 M colchicine produced approximately a 2-fold increase in collagenase in the culture medium, a finding shown by biosynthetic studies to be attributable to enhanced synthesis of enzyme protein. In the case of typical **recessive dystrophic epidermolysis bullosa**, a disease characterized by excessive collagenase synthesis, the fibroblasts could also be stimulated to produce additional collagenase, despite having elevated baseline synthetic rates. Fibroblasts isolated from 1 recessive epidermolysis bullosa [REB] patient were resistant to the stimulatory effects of colchicine in concentrations up to 5 .times. 10-6 M. In the absence of colchicine, collagenase synthesis in this patient's cells (termed REBc-) was 3-4 times that of normal controls, suggesting that the as yet undefined cellular function that is abrogated (or stimulated) by colchicine in normal cells may have been genetically impaired in these REBc- cells. Despite the resistance to colchicine, as manifested by the failure to stimulate collagenase, gross parameters of microtubular function, such as cell replication, were intact. Phenotypically, this patient had a form of epidermolysis bullosa intermediate between typical recessive dystrophic and recessive letalis forms of the disease. Although an experimentally induced blister was located in the lamina lucida, hypoplastic anchoring fibrils were also observed. These findings, in

addition to the marked increase in collagenase synthesis, suggest the possibility that this patient may represent a compound heterozygote of 2 forms of epidermolysis bullosa and that colchicine may be useful in defining other such patients.

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L5 ANSWER 52 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on curcumin and curcuminoids. Part 18. Evaluation of Curcuma products by the use of standardized reference color values
SO Zeitschrift fuer Lebensmittel-Untersuchung und -Forschung (1992), 194(2), 129-30
CODEN: ZLUFAR; ISSN: 0044-3026
AB Studies of com. prepns. of "pure" curcumin (I) by the author's HPLC method showed the presence of 7.1-17.2% of demethoxy-I and 1.0-9.6% of bis-demethoxy-I. Evaluation of these contents in the same samples by absorbance measurements, and consideration of the author's own color values (CV) for the pure compds. measured at 420, 425, and 430 nm, showed agreement to within 5.8%. The necessity of using accurate CV measured with pure ref. materials when quantifying the food dye I in com. prepns. by absorbance measurements is underlined.
PY 1992
AU Toennesen, Hanne Hjorth

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L5 ANSWER 55 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on curcumin and **curcuminoids**. XVI. Effect of curcumin analogs on hyaluronic acid degradation in vitro
SO International Journal of Pharmaceutics (1989), 51(3), 259-61
CODEN: IJPHDE; ISSN: 0378-5173
AB Curcumin, **demethoxycurcumin**, and bisdemethoxycurcumin of Curcuma longa showed a catalytic effect on the degrdn. of hyaluronic acid (HA) by influencing the formation of OH radical which caused the HA depolymn. The effect of **curcuminoids** was inhibited by addn. of a hydroxyl radical quencher (mannitol).
PY 1989
AU Toennesen, Hanne Hjorth

L5 ANSWER 56 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on curcumin and **curcuminoids**. XV. Catalytic effect of demethoxy- and bisdemethoxycurcumin on the peroxidation of linoleic acid by 15-lipoxygenase
SO International Journal of Pharmaceutics (1989), 51(2), 179-81
CODEN: IJPHDE; ISSN: 0378-5173
AB Curcumin, **demethoxycurcumin**, and bisdemethoxycurcumin (0.0067-0.0003 mg/mL) had a catalytic effect on 15-lipoxygenase-mediated peroxidn. of linoleic acid. Bisdemethoxycurcumin at 0.0133 mg/mL had an inhibitory action. The com. prepn. contg. all 3 curcumins had a synergistic action. The Lineweaver plots for demethoxy- and bisdemethoxycurcumin indicated an uncompetitive activation mechanism. The anti-inflammatory action of curcumins is discussed in light of the above findings.
PY 1989
AU Toennesen, Hanne Hjorth

L5 ANSWER 57 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI High performance liquid chromatographic analysis of **curcuminoids** and their photo-oxidative decomposition compounds in Curcuma longa L
SO Journal of Liquid Chromatography (1988), 11(11), 2295-304
CODEN: JLCHD8; ISSN: 0148-3919
AB Photochem. oxidn. of **curcuminoids** such as curcumin, bisdemethoxycurcumin, and **demethoxycurcumin** in dry powder of Curcuma longa (zingiberaceae) root and in EtOH and MeOH exts. has been studied following sunlight exposure for 120 h. Whatman PartiSphere-5 NH2 and Whatman PartiSphere-5 WCX columns were used to analyze **curcuminoids** and their degrdn. products. The **curcuminoids** were found to be more stable in the dry powder of C. longa root than in EtOH and MeOH exts. Vanillin, p-hydroxybenzaldehyde, ferulic aldehyde, p-hydroxybenzoic acid, vanillic acid, and ferulic acid were identified as the oxidn. products.
PY 1988
AU Khurana, Amrik; Ho, Chi Tang

L5 ANSWER 58 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI High performance liquid chromatographic separation and spectral characterization of the pigments in turmeric and annatto
SO Journal of Food Science (1988), 53(6), 1823-6
CODEN: JFDSAZ; ISSN: 0022-1147
AB High performance liq. chromatog. sepns. for the detn. of the pigments in the food colorants, annatto and turmeric, were developed. Chromatog. anal. time for the isocratic system was 10 min and 22 min for the gradient system. The isocratic sepn. employed a 25 cm .times. 4.6 mm i.d. Zorbax ODS column with a 58/42 (vol./vol.) mixt. of water/tetrahydrofuran, THF,

at 1 mL/min. A gradient water/THF system using the same components was also developed to improve the resoln. between the **curcuminoids**. Sample prepn. consists of diln. and filtration of the exts. The 3 major pigments in turmeric, identified from visible and fluorescence stop flow spectra, were curcumin, **demethoxycurcumin**, and bisdemethoxycurcumin. Absorbance, excitation and emission max. for the 3 **curcuminoids** plus the carotenoids, bixin and norbixin, were detd.

PY 1988
AU Rouseff, Russell L.

L5 ANSWER 59 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Separation and determination of **curcuminoids** in Curcuma longa L. and its preparation by HPLC
SO Yaoxue Xuebao (1986), 21(5), 382-5
CODEN: YHHPAL; ISSN: 0513-4870
AB Curcumin (I) [458-37-7], **demethoxycurcumin** (II) [22608-11-3] and bisdemethoxycurcumin (III) were detd. in C. longa by HPLC by using a YWG-C18H35 column, THF-H2O-HOAC (36:58:6 vol./vol./vol.) as the mobile phase, and detector set at 254 nm. Naphthalene was used as internal std. Recoveries were 96.2-96.9% and the relative std. deviations 1.74, 0.67, and 0.53% for I, II, and III, resp. The method is fast and simple with no interference.
PY 1986
AU Zhao, Deyong; Yang, Mokun

L5 ANSWER 60 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on Chinese Curcuma plants. IV. Assay of **curcuminoids** in the root and tuber of Curcuma spp
SO Zhongcaoyao (1983), 14(2), 59-63
CODEN: CTYAD8; ISSN: 0253-2670
AB curcumin (I) [458-37-7], **demethoxycurcumin** (II) and bis(demethoxy)curcumin (III) were detd. in the root or bulb of Curcuma by spectrophotometry. The root or bulb was dried, powd. and the powd was extd. into MeOH, which was dild. and analyzed at 418 nm for the detn. of total **curcuminoids**. For the detn. of I, II and III, the ext. was sepd. by TLC and fractions were measured spectrophotometrically at 428, 422 and 417 nm, resp. The calibration plots were linear to .apprx.4 .mu.g/mL. Highest **curcuminoid** contents were detected in C. longa, followed by C. wenyujin, C. xanthorrhiza, C. kwangsiensis and C. aeruginosa. Regional and seasonal variations in the **curcuminoid** contents were obsd.
PY 1983
AU Chen, Jianmin; Chen, Yuheng; Yu, Jingguang

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L5 ANSWER 50 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on curcumin and **curcuminoids**. Part 21. Variation in the **curcuminoid** content in Curcuma longa and C. aromatica from India during one season
SO Zeitschrift fuer Lebensmittel-Untersuchung und -Forschung (1992), 194(6), 570-2
CODEN: ZLUFAR; ISSN: 0044-3026
AB **Curcuminoid** levels in the primary and secondary rhizomes (bulbs and fingers) of 3 C. longa and 3 C. aromatica varieties decreased during a 17-wk maturity period (between age 19-36 wks). Although the total pigment levels of both species were similar, a slight difference in curcumin and demethoxy- and bisdemethoxycurcumin distribution between the rhizome parts

was obsd.
PY 1992
AU Toennesen, Hanne Hjorth; Karlsen, Jan; Grislingaas, Anne Lise; Balakrishnan, Korattiyil Velayudhan Nair; Ayyappan, Payyeri; Verghese, James

L5 ANSWER 51 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Antioxidant activity of tropical ginger extracts and analysis of the contained **curcuminoids**
SO Journal of Agricultural and Food Chemistry (1992), 40(8), 1337-40
CODEN: JAFCAU; ISSN: 0021-8561
AB Antioxidant activities of the rhizomes of 9 tropical gingers (Curcuma aeruginosa, C. domestica, C. heyneana, C. mangga, C. xanthorrhiza, Zingiber cassumunar, Phaeomeria speciosa, Alpinia galanga, and Amomum kepulaga) were measured by thiocyanate and TBA methods in a aq. alc. system after extn. and fractionation with org. solvents. The quantity of 3 known **curcuminoids** (curcumin, **demethoxycurcumin** and bisdemethoxycurcumin), potent antioxidants of ginger and spice species, in the exts. was detd. by HPLC. The antioxidant activity of the spice exts. was greater than that estd. from the actual quantity of 3 known **curcuminoids** in the exts.
PY 1992
AU Jitoe, Akiko; Masuda, Toshiya; Tengah, I. G. P.; Suprapta, Dewa N.; Gara, I. W.; Nakatani, Nobuji

L5 ANSWER 52 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on curcumin and **curcuminoids**. Part 18. Evaluation of Curcuma products by the use of standardized reference color values
SO Zeitschrift fuer Lebensmittel-Untersuchung und -Forschung (1992), 194(2), 129-30
CODEN: ZLUFAR; ISSN: 0044-3026
AB Studies of com. prepns. of "pure" curcumin (I) by the author's HPLC method showed the presence of 7.1-17.2% of demethoxy-I and 1.0-9.6% of bis-demethoxy-I. Evaluation of these contents in the same samples by absorbance measurements, and consideration of the author's own color values (CV) for the pure compds. measured at 420, 425, and 430 nm, showed agreement to within 5.8%. The necessity of using accurate CV measured with pure ref. materials when quantifying the food dye I in com. prepns. by absorbance measurements is underlined.
PY 1992
AU Toennesen, Hanne Hjorth

L5 ANSWER 53 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on curcumin and **curcuminoids**. Part 19. Evaluation of thin-layer chromatography for the quantitation of curcumin and **curcuminoids**
SO Zeitschrift fuer Lebensmittel-Untersuchung und -Forschung (1991), 193(6), 548-50
CODEN: ZLUFAR; ISSN: 0044-3026
AB TLC was evaluated as an alternative to HPLC for the quantitation of **curcuminoids** in Curcuma exts. Thus, curcumin (I) was effectively sepd. from demethoxy-I or bisdemethoxy-I on either pure silica gel or an amino-bonded gel, although improved I stability and lower irreversible adsorption at the application spot were obsd. with the latter phase. A std. deviation of .ltoreq.6% was obsd. for all **curcuminoid** detns. at the concn. range 0.008-0.08 mg/mL for an application vol. of 10 .mu.L to the amino phase compared to HPLC, indicating the former a viable alternative providing conditions and approx. starting conditions are standardized.

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PY 1991
AU Toennesen, Hanne Hjorth; Grislingaas, Anne Lise; Karlsen, Jan

L5 ANSWER 54 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on curcumin and **curcuminoids**. Part 17. Variation in the content of **curcuminoids** in Curcuma longa from Nepal during one season
SO Zeitschrift fuer Lebensmittel-Untersuchung und -Forschung (1989), 189(2), 116-18
CODEN: ZLUFAR; ISSN: 0044-3026
AB The av. **curcuminoid** content of the rhizomes of C. longa included 1.11, 0.86, and 1.62% of curcumin (I), demethoxy-I, and bis-demethoxy-I, resp.; no changes in these levels were obsd. during a 17-wk. growth period.
PY 1989
AU Toennesen, Hanne Hjorth; Karlsen, Jan; Adhikary, Sitaram R.; Pandey, Rita

L5 ANSWER 55 OF 60 CAPLUS COPYRIGHT 2003 ACS on STN
TI Studies on curcumin and **curcuminoids**. XVI. Effect of curcumin analogs on hyaluronic acid degradation in vitro
SO International Journal of Pharmaceutics (1989), 51(3), 259-61
CODEN: IJPHDE; ISSN: 0378-5173
AB Curcumin, **demethoxycurcumin**, and bisdemethoxycurcumin of Curcuma longa showed a catalytic effect on the degrdn. of hyaluronic acid (HA) by influencing the formation of OH radical which caused the HA depolymn. The effect of **curcuminoids** was inhibited by addn. of a hydroxyl radical quencher (mannitol).
PY 1989
AU Toennesen, Hanne Hjorth

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L4 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2003 ACS on STN
TI Microarray-based analysis of anti-angiogenic activity of **demethoxycurcumin** on human umbilical vein endothelial cells: Crucial involvement of the down-regulation of matrix metalloproteinase
SO Japanese Journal of Cancer Research (2002), 93(12), 1378-1385
CODEN: JJCREP; ISSN: 0910-5050
AB CDNA microarray-based gene expression anal. has been successfully employed to explore the action mechanism and to validate the targets of several drugs. In the present study, we evaluated anti-angiogenic activity of **demethoxycurcumin** (DC), a structural analog of **curcumin**, isolated from Curcuma aromatica, and investigated the effect of DC on genetic reprogramming in cultured human umbilical vein endothelial cells (HUVECs) using cDNA microarray anal. Of 1024 human cancer-focused genes arrayed, 187 genes were up-regulated and 72 genes were down-regulated at least 2-fold by DC. Interestingly, 9 **angiogenesis**-related genes were down-regulated over 5-fold in response to DC, suggesting that the genetic reprogramming was crucially involved in anti-**angiogenesis** by the compd. To verify the results obtained from cDNA microarray anal., matrix metalloproteinase-9 (MMP-9), the product of one of the **angiogenesis**-related genes down-regulated over 5-fold by DC, was investigated using gelatin zymog. DC potently inhibited the expression of MMP-9, yet showed no direct effect on its activity. These data show that gene expressional change of MMP-9 is a major mediator for **angiogenesis** inhibition by DC.
PY 2002
AU Kim, Jin Hee; Shim, Joong Sup; Lee, Seok-Ki; Kim, Kyu-Won; Rha, Sun Young; Chung, Hyun Cheol; Kwon, Ho Jeong

L4 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2003 ACS on STN
TI Hydrazinocurcumin, a novel synthetic **curcumin** derivative, is a potent inhibitor of endothelial cell proliferation
SO Bioorganic & Medicinal Chemistry (2002), 10(8), 2439-2444
CODEN: BMECEP; ISSN: 0968-0896
AB **Curcumin** and some of its derivs. were known as in vivo inhibitors of **angiogenesis**. In present study, a novel **curcumin** deriv., named hydrazinocurcumin (HC) was synthesized and examd. for its biol. activities. HC potently inhibited the proliferation of bovine aortic endothelial cells (BAECs) at a nanomolar concn. (IC50=520 nM) without cytotoxicity. In vivo and in vitro **angiogenesis** expts. showed HC as a new candidate for anti-angiogenic agent.
PY 2002
AU Sup Shim, Joong; Hoon Kim, Dong; Jung, Hye Jin; Hee Kim, Jin; Lim, Dongyeol; Lee, Seok-Ki; Kim, Kyu-Won; Ahn, Jong Woong; Yoo, Jong-Shin; Rho, Jung-Rae; Shin, Jongheon; Jeong Kwon, Ho

L4 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2003 ACS on STN
TI **Curcumin** and **curcuminoid** inhibition of **angiogenesis**
SO PCT Int. Appl., 30 pp.
CODEN: PIXXD2
AB Methods for treating diseases or disorders of the skin which are characterized by **angiogenesis** have been developed using **curcumin** and **curcumin** analogs. Based on the results obtained with **curcumin**, it has been detd. that other **angiogenesis** inhibitors can also be used to treat these skin disorders. It has further been discovered that **curcumin** acts to inhibit **angiogenesis** in part by inhibition of basic fibroblast growth factor (bFGF), and thereby provides a means for treating other

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disorders characterized by elevated levels of bFGF, such as bladder cancer, using **curcumin** and other analogs which also inhibit bFGF. Representative skin disorders to be treated include the malignant diseases angiosarcoma, hemangioendothelioma, basal cell carcinoma, squamous cell carcinoma, malignant melanoma and Kaposi's sarcoma, and the non-malignant diseases or conditions including psoriasis, lymphangiogenesis, hemangioma of childhood, Sturge-Weber syndrome, verruca vulgaris, neurofibromatosis, tuberous sclerosis, pyogenic granulomas, recessive dystrophic epidermolysis bullosa, venous ulcers, acne, rosacea, eczema, molluscum contagious, seborrheic keratosis, and actinic keratosis.

PY 2001
2002
2002
2001

IN Arbiser, Jack L.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001000201	A1	20010104	WO 2000-US17608	20000627
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 2002006966	A1	20020117	US 1999-345712	19990630
EP 1196158	A1	20020417	EP 2000-941736	20000627
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 2001025034	A1	20010927	US 2001-765491	20010118

L4 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2003 ACS on STN

TI **Curcuminoids** inhibit the angiogenic response stimulated by fibroblast growth factor-2, including expression of matrix metalloproteinase gelatinase B

SO Journal of Biological Chemistry (2000), 275(14), 10405-10412
CODEN: JBCHA3; ISSN: 0021-9258

AB We have studied mechanisms controlling activation of the gelatinase B gene (matrix metalloproteinase-9) by fibroblast growth factor-2 (FGF-2) during **angiogenesis**, and the effects of the natural product **curcuminoids** on this process. Using a transgenic mouse (line 3445) harboring a gelatinase B promoter/lacZ fusion gene, we demonstrate FGF-2 stimulation of reporter gene expression in endothelial cells of invading neocapillaries in the corneal micropocket assay. Using cultured corneal cells, we show that FGF-2 stimulates DNA binding activity of transcription factor AP-1 but not NF-.kappa.B and that AP-1 stimulation is inhibited by **curcuminoids**. We further show that induction of gelatinase B transcriptional promoter activity in response to FGF-2 is dependent on AP-1 but not NF-.kappa.B response elements and that promoter activity is also inhibited by **curcuminoids**. In rabbit corneas, the angiogenic response induced by implantation of an FGF-2 pellet is inhibited by the coimplantation of a **curcuminoid** pellet, and this correlates with inhibition of endogenous gelatinase B expression induced by FGF-2. Angiostatic efficacy in the cornea is also, obsd. when **curcuminoids** are provided to mice in the diet. Our findings provide evidence that **curcuminoids** target the FGF-2 angiogenic signaling pathway and inhibit expression of gelatinase B in the angiogenic process.

PY 2000

AU Mohan, Royce; Sivak, Jeremy; Ashton, Paul; Russo, Laoti A.; Pham, Bao Q.; Kasahara, Niro; Raizman, Michael B.; Fini, M. Elizabeth

L4 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2003 ACS on STN

Paddy

TI **Curcumin** is an in vivo inhibitor of **angiogenesis**
SO Molecular Medicine (New York) (1998), 4(6), 376-383
CODEN: MOMEF3; ISSN: 1076-1551
AB **Curcumin** is a small-mol.-wt. compd. that is isolated from the commonly used spice turmeric. In animal models, **curcumin** and its derivs. have been shown to inhibit the progression of chem. induced colon and skin cancers. The genetic changes in carcinogenesis in these organs involve different genes, but **curcumin** is effective in preventing carcinogenesis in both organs. A possible explanation for this finding is that **curcumin** may inhibit **angiogenesis**.
Curcumin was tested for its ability to inhibit the proliferation of primary endothelial cells in the presence and absence of basic fibroblast growth factor (bFGF), as well as its ability to inhibit proliferation of an immortalized endothelial cell line. **Curcumin** and its derivs. were subsequently tested for their ability to inhibit bFGF-induced corneal neovascularization in the mouse cornea. Finally, **curcumin** was tested for its ability to inhibit phorbol ester-stimulated vascular endothelial growth factor (VEGF) mRNA prodn. **Curcumin** effectively inhibited endothelial cell proliferation in a dose-dependent manner. **Curcumin** and its derivs. demonstrated significant inhibition of bFGF-mediated corneal neovascularization in the mouse. **Curcumin** had no effect on phorbol ester-stimulated VEGF prodn. These results indicate that **curcumin** has direct antiangiogenic activity in vitro and in vivo. The activity of **curcumin** in inhibiting carcinogenesis in diverse organs such as the skin and colon may be mediated in part through **angiogenesis** inhibition.
PY 1998
AU Arbiser, Jack L.; Klauber, Nancy; Rohan, Richard; Van Leeuwen, Robert; Huang, Mou-Tuan; Fisher, Carolyn; Flynn, Evelyn; Byers, H. Randolph

Paddy

L7 ANSWER 6 OF 6 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AN 97:32048 SCISEARCH
GA The Genuine Article (R) Number: VZ915
TI The prognostic significance of **basic fibroblast growth factor** in **cutaneous malignant melanoma**
AU AlAlousi S; Barnhill R (Reprint); Blessing K; Barksdale S
CS HARVARD UNIV, DEPT PATHOL, DIV DERMATOPATHOL, BRIGHAM & WOMENS HOSP, SCH MED, 75 FRANCIS ST, BOSTON, MA 02115 (Reprint); HARVARD UNIV, DEPT PATHOL, DIV DERMATOPATHOL, BRIGHAM & WOMENS HOSP, SCH MED, BOSTON, MA 02115; UNIV ABERDEEN, ABERDEEN, SCOTLAND
CYA USA; SCOTLAND
SO JOURNAL OF CUTANEOUS PATHOLOGY, (DEC 1996) Vol. 23, No. 6, pp. 506-510. Publisher: MUNKSGAARD INT PUBL LTD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK.
ISSN: 0303-6987.
DT Article; Journal
FS CLIN
LA English
REC Reference Count: 11
AB Basic fibroblast growth factor (bFGF) is a growth factor and an angiogenesis factor which may play a role in the evolution of cutaneous malignant melanoma (CMM). In this study, we evaluated the distribution of bFGF in CMM using immunochemical methods and correlated the pattern of bFGF expression with the clinical course. Formalin-fixed, paraffin-embedded sections of 46 CMMs were immunostained with a high-affinity purified antibody raised against human bFGF. CMM were categorized into lesions that exhibited subsequent recurrence (local, regional and/or systemic) or recurrence-free lesions. The minimum follow-up time was 5 years. Expression of bFGF within the tumors and in peritumoral and intratumoral blood vessels was similar in the two groups. Comparable results were attained when 8 recurring vs 8 non-recurring CMM, selected from the above tumors, were matched for age, gender, anatomic site and tumor thickness. These results suggest that the biologic behavior of CMM may not be predicted by immunoreactivity to bFGF in CMM cells or in the local tumor vasculature. (C) Munksgaard 1996.
CC PATHOLOGY; DERMATOLOGY & VENEREAL DISEASES
STP KeyWords Plus (R): MELANOCYTIC LESIONS; HYBRIDIZATION; LOCALIZATION
RF 95-2001 001; BASIC FIBROBLAST GROWTH-FACTOR; VASCULAR SMOOTH-MUSCLE CELLS; EXPRESSION OF THE BASEMENT-MEMBRANE HEPARAN-SULFATE PROTEOGLYCAN (PERLECAN)
RE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)
ALOUSI S A	1996	23	118	J CUTAN PATHOL
FLEMING M G	1992	14	496	AM J DERMATOPATH
GOSPODAROWICZ D	1990	114	15	UCLA S MOL CELLULAR
HALABAN R	1991	10	129	CANCER METAST REV
KLAGSMAN M	1991		229	CELL
KORHONEN J	1992		91	ANGIOGENESIS KEY PRI
REED J A	1994	144	329	AM J PATHOL
SCHULZEOSTHOFF K	1990	137	85	AM J PATHOL
SCOTT G	1991	96	318	J INVEST DERMATOL
VLODAVSKY I	1987	84	2292	P NATL ACAD SCI USA
YAYON A	1990	9	191	CANCER METAST REV

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L15 ANSWER 10 OF 21 DGENE COPYRIGHT 2003 THOMSON DERWENT on STN
AN AAQ10791 DNA DGENE
TI DNA encoding vascular endothelial cell growth factor - used for producing the factor for angiogenesis and re-endothelialisation in wound healing
IN Tischer E R; Abrhamam; Fiddes J C; Mitchell R L
PA (CALD) CALIFORNIA BIOTECHNOLOGY INC.
PI **WO 9102058 A 19910221 94p**
AI WO 1990-US4227 19900727
PRAI US 1989-450883 19891214
US 1989-387545 19890727
DT Patent
LA English
OS 1991-073534 [10]
CR P-PSDB: AAR10911
DESC Bovine vascular endothelial cell growth factor 164.
PI **WO 9102058 A 19910221 94p**
AB . . . AAQ10796 for bVEGF120 obtained by alternative splicing this sequence, i.e. bases 342-473 are spliced. The product can be used for **angiogenesis** and re-endothelialisation of inner vascular surfaces in wound healing, e.g. treatment of full- thickness wounds such as dermal **ulcers**, **venous ulcers** and diabetic **ulcers**, burns, in surgery, in balloon angioplasty and for the in vitro culturing of endothelial cells. Hybrid growth factors of PDGF. . . VEGF can exhibit a mitogenic profile between each factor and can be used for wound healing or as inhibitors of **angiogenesis** for e.g. preventing the growth of tumours. VEGF analogues in which CYS residues are substd. are more stable. See also. . .

(FILE 'HOME' ENTERED AT 13:11:50 ON 31 OCT 2003)

FILE 'ADISCTI, ADISINSIGHT, ADISNEWS, BIOSIS, BIOTECHNO, CANCERLIT, CAPLUS, CEN, DISSABS, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, DRUGU, EMBAL, EMBASE, ESBIOBASE, IFIPAT, IPA, JICST-EPLUS, KOSMET, LIFESCI, MEDICONF, MEDLINE, NAPRALERT, NLDB, NUTRACEUT, ..' ENTERED AT 13:12:06 ON 31 OCT 2003

L1	1602	S	RECESSIVE DYSTROPHIC EPIDERMOLYSIS BULLOSA
L2	824	S	L1 AND PD<2000
L3	0	S	L2 AND (ANGIOGENESIS ANTIANGIOGENIC OR ANTIANGIOSTATIC)
L4	0	S	L3 AND (BASIC AND FIBROBLAST)
L5	27	S	L1 AND (ANGIO? OR ANGIOGENESIS OR ANGIOSTATIC OR ANTIANGIOGE
L6	13		DUP REM L5 (14 DUPLICATES REMOVED)
L7	1359	S	VENOUS (W) ULCER
L8	85	S	L7 AND (ANGIOGENESIS OR ANTIANGIOGENIC OR ANGIOSTATIC)
L9	10	S	L8 AND PD<2000
L10	1	S	L9 AND (FIBROBLAST)/AB
L11	2255	S	VENOUS (P) ANGIOGENESIS
L12	435	S	L11 AND PD<2000
L13	50	S	L12 AND (ULCER OR ULCERS)
L14	34		DUP REM L13 (16 DUPLICATES REMOVED)
L15	21	S	L14 AND (VENOUS (W) (ULCER OR ULCERS))

(FILE 'HOME' ENTERED AT 13:11:50 ON 31 OCT 2003)

FILE 'ADISCTI, ADISINSIGHT, ADISNEWS, BIOSIS, BIOTECHNO, CANCERLIT, CAPLUS, CEN, DISSABS, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, DRUGU, EMBAL, EMBASE, ESBIOBASE, IFIPAT, IPA, JICST-EPLUS, KOSMET, LIFESCI, MEDICONF, MEDLINE, NAPRALERT, NLDB, NUTRACEUT, ..' ENTERED AT 13:12:06 ON 31 OCT 2003

L1	1602	S	RECESSIVE DYSTROPHIC EPIDERMOLYSIS BULLOSA
L2	824	S	L1 AND PD<2000
L3	0	S	L2 AND (ANGIOGENESIS ANTIANGIOGENIC OR ANTIANGIOSTATIC)
L4	0	S	L3 AND (BASIC AND FIBROBLAST)
L5	27	S	L1 AND (ANGIO? OR ANGIOGENESIS OR ANGIOSTATIC OR ANTIANGIOGE
L6	13		DUP REM L5 (14 DUPLICATES REMOVED)
L7	1359	S	VENOUS (W) ULCER
L8	85	S	L7 AND (ANGIOGENESIS OR ANTIANGIOGENIC OR ANGIOSTATIC)
L9	10	S	L8 AND PD<2000
L10	1	S	L9 AND (FIBROBLAST)/AB
L11	2255	S	VENOUS (P) ANGIOGENESIS
L12	435	S	L11 AND PD<2000
L13	50	S	L12 AND (ULCER OR ULCERS)
L14	34		DUP REM L13 (16 DUPLICATES REMOVED)
L15	21	S	L14 AND (VENOUS (W) (ULCER OR ULCERS))
L16	1	S	L7 AND (CURCUMIN OR CURCUMINOID OR DEMETHOXYCURCUMIN)

L3 ANSWER 10 OF 27 USPATFULL on STN
AN 97:3837 USPATFULL
TI Methods and compositions for inhibition of angiogenesis
IN D'Amato, Robert, Lancaster, PA, United States
PA The Children's Medical Center Corporation, Boston, MA, United States (U.S. corporation)
PI US 5593990 19970114 <--
AI US 1995-371987 19950113 (8)
RLI Continuation-in-part of Ser. No. US 1993-168817, filed on 15 Dec 1993 which is a continuation-in-part of Ser. No. US 1993-25046, filed on 1 Mar 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Jordan, Kimberly
LREP Jones & Askew
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 895
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
PI US 5593990 19970114 <--
SUMM One example of a disease mediated by **angiogenesis** is ocular neovascular disease. This disease is characterized by invasion of new blood vessels into the structures of the eye. . . limited to, epidemic keratoconjunctivitis, Vitamin A deficiency, contact lens overwear, atopic keratitis, superior limbic keratitis, pterygium keratitis sicca, sjogrens, acne **rosacea**, phylectenulosis, syphilis, Mycobacteria infections, lipid degeneration, chemical burns, bacterial ulcers, fungal ulcers, Herpes simplex infections, Herpes zoster infections, protozoan infections,. . .

L8 ANSWER 28 OF 32 USPATFULL on STN
PI US 5190918 19930302 <--
DETD "**Angiogenesis** activity" is defined herein as the ability to inhibit or enhance the formation of blood vessels or **lymph** vessels.
DETD **Angiogenesis** is the formation of blood and **lymph** vessels. The compounds of this invention are useful in the modulation of **angiogenesis**, particularly in enhancing wound healing, inhibiting or preventing tumor growth, diabetic retinopathy, and rheumatoid arthritis. Standard **angiogenesis** assays are well known in the art.

L8 ANSWER 17 OF 32 USPATFULL on STN
PI US 5639725 19970617 <--
SUMM As used herein, the term "**angiogenesis**" means the generation of new blood vessels into a tissue or organ. Under normal physiological conditions, humans or animals undergo **angiogenesis** only in very specific restricted situations. For example, **angiogenesis** is normally observed in wound healing, fetal and embryonal development and formation of the corpus luteum, endometrium and placenta. The term "endothelium" means a thin layer of flat epithelial cells that lines serous cavities, **lymph** vessels, and blood vessels. The term "endothelial inhibiting activity" means the capability of a molecule to inhibit **angiogenesis** in general and, for example, to inhibit the growth of bovine capillary endothelial cells in culture in the presence of. . .

L7 ANSWER 1 OF 3 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 1
AN 1998424010 EMBASE
TI Phacomatosis pigmentovascularis type IIb associated with **Sturge-Weber syndrome** and pyogenic granuloma.
AU Hagiwara K.; Uezato H.; Nonaka S.
CS Dr. K. Hagiwara, Department of Dermatology, Research Ctr. of Comprehensive Med., University of tile Ryukyus, 207 Uehara, Nishihara, Okinawa 903-0215, Japan
SO Journal of Dermatology, (**1998**) 25/11 (721-729).
Refs: 26
ISSN: 0385-2407 CODEN: JDMYAG
CY Japan
DT Journal; Article
FS 007 Pediatrics and Pediatric Surgery
013 Dermatology and Venereology
LA English
SL English
TI Phacomatosis pigmentovascularis type IIb associated with **Sturge-Weber syndrome** and pyogenic granuloma.
SO Journal of Dermatology, (**1998**) 25/11 (721-729).
Refs: 26
ISSN: 0385-2407 CODEN: JDMYAG
AB A case of phacomatosis pigmentovascularis (PPV) in a 6-year-old girl with **Sturge-Weber syndrome**, pyogenic granuloma, and other complications is described. It is relatively rare that a complete form of **Sturge-Weber syndrome** was associated with PPV. A review of the literature on PPV, focusing on total number of reported cases and etiological. . . skin, 6.85 .+-. 4.9/mm2 (n=20). There was a significant difference between the two, indicating that MCs are closely associated with **angiogenesis** in pyogenic granuloma.
CT Medical Descriptors:
*phakomatosis: CN, congenital disorder
*phakomatosis: DI, diagnosis
***Sturge Weber syndrome**
*pyogenic granuloma
disease association
mast cell
angiogenesis
skin hemangioma
nevus flammeus
capillary hemangioma
skin manifestation
human
female
case report
child
article
*tryptase: EC, endogenous compound

L7 ANSWER 2 OF 3 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 2
AN 96094511 EMBASE
DN 1996094511
TI Axial structures control laterality in the distribution pattern of endothelial cells.
AU Klessinger S.; Christ B.
CS Institute of Anatomy, University of Freiburg, PO Box 111,D-79001 Freiburg, Germany
SO Anatomy and Embryology, (**1996**) 193/4 (319-330).
ISSN: 0340-2061 CODEN: ANEMDG
CY Germany
DT Journal; Article

FS 001 Anatomy, Anthropology, Embryology and Histology
021 Developmental Biology and Teratology
LA English
SL English
SO Anatomy and Embryology, (**1996**) 193/4 (319-330).
ISSN: 0340-2061 CODEN: ANEMDG
AB . . . substances. It is conceivable that our results can explain the lateralization of illnesses of the vascular system, as the Klippel-Trenaunay **syndrome** or the **Sturge-Weber syndrome**.
CT Medical Descriptors:
***angiogenesis**
*cell migration
*embryo axis
*endothelium cell
animal cell
article
embryo
histology
mesoderm
nonhuman
priority journal
quail

L7 ANSWER 3 OF 3 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 95062538 EMBASE
DN 1995062538
TI Expression of basement membrane and endothelial cell adhesion molecules in vascular malformations of the brain: Preliminary observations and working hypothesis.
AU Robinson Jr. J.R.; Awad I.A.; Zhou P.; Barna B.P.; Estes M.L.
CS Barrow Neurological Institute, 350 West Thomas Road,Phoenix, AZ 85013-4496, United States
SO Neurological Research, (**1995**) 17/1 (49-58).
ISSN: 0161-6412 CODEN: NRESDZ
CY United Kingdom
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LA English
SL English
SO Neurological Research, (**1995**) 17/1 (49-58).
ISSN: 0161-6412 CODEN: NRESDZ
AB . . . We have freeze-processed four specimens of arteriovenous malformation (AVM), two cavernous malformations (CM), and resected cortex from one case of **Sturge-Weber** disease (SWD) for immunohistochemical studies. Probes of vascular maturity and cellular adhesion were examined, including Factor 8 related antigen (F8RAG),. . .
CT Medical Descriptors:
*brain arteriovenous malformation: CN, congenital disorder
*brain arteriovenous malformation: ET, etiology
*cavernous hemangioma: ET, etiology
*cavernous hemangioma: CN, congenital disorder
***sturge weber syndrome: CN, congenital disorder**
***sturge weber syndrome: ET, etiology**
angiogenesis
antigen expression
article
basement membrane
cell adhesion
clinical article
controlled study
endothelium cell

human
human tissue
protein localization
*cell adhesion molecule: EC, endogenous compound
blood clotting factor 8: EC, endogenous compound
endothelial. . .

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L17 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS on STN
AN 1991:604988 CAPLUS
DN 115:204988
TI Increase in .alpha.-actin and basic fibroblast growth factor in angiofibromas of patients with tuberous sclerosis
AU Takanashi, Masanori; Nakayama, Juichiro; Inoue, Mitsuse; Urabe, Atsumichi; Hori, Yoshiaki
CS Sch. Med., Kyushu Univ., Fukuoka, 812, Japan
SO Nippon Hifuka Gakkai Zasshi (**1991**), 101(6), 601-8
CODEN: NHKZAD; ISSN: 0021-499X
DT Journal
LA Japanese
CC 14-10 (Mammalian Pathological Biochemistry)
AB There was an increase of .alpha.-actin-pos. microvessels in the papilla and the upper reticular dermis of 4 angiofibromas and a connective tissue nevus in **tuberous sclerosis** patients compared to those of normal skin. .alpha.-Actin in the microvessels localized mainly in pericytes. Most of the basic fibroblast growth factor-pos. microvessels corresponded to those contg. .alpha.-actin as detd. by double immunostaining. Evidently, the increased basic fibroblast growth factor plays a role in stimulating **angiogenesis** and/or maintaining vessels in angiofibromas.
ST tuberous sclerosis alpha actin skin angiofibroma; basic fibroblast growth factor actin angiofibroma
IT Skin, neoplasm
(angiofibroma, .alpha.-actin and basic fibroblast growth factor of, in tuberous sclerosis of humans)
IT Neoplasm, composition
(nevus, of skin, .alpha.-actin and basic fibroblast growth factor of, in tuberous sclerosis of humans)
IT Fibroma
(angio-, .alpha.-actin and basic fibroblast growth factor of, of skin, in tuberous sclerosis of humans)
IT Skin, neoplasm
(nevus, .alpha.-actin and basic fibroblast growth factor of, in tuberous sclerosis of humans)
IT Brain, disease or disorder
(tuberous sclerosis, .alpha.-actin and basic fibroblast growth factor of skin angiofibroma in, of humans)
IT Actins
RL: BIOL (Biological study)
(.alpha.-, of angiofibroma of skin, in tuberous sclerosis of humans, basic fibroblast growth factor in relation to)
IT 106096-93-9, Basic fibroblast growth factor
RL: BIOL (Biological study)
(of angiofibroma of skin, in tuberous sclerosis of humans, .alpha.-actin in relation to)

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(FILE 'HOME' ENTERED AT 06:18:23 ON 31 OCT 2003)

FILE 'ADISCTI, ADISINSIGHT, ADISNEWS, BIOSIS, CANCERLIT, CAPLUS, CEN, DISSABS, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, DRUGU, EMBAL, EMBASE, ESBIOBASE, IFIPAT, IPA, JICST-EPLUS, KOSMET, LIFESCI, MEDICONF, MEDLINE, NAPRALERT, NLDB, NUTRACEUT, PASCAL, ...' ENTERED AT 06:18:35 ON 31 OCT 2003

L1	15659	S (STURGE AND WEBER AND SYNDROME)
L2	12373	S L1 AND ANGIOGENESIS
L3	5	S L2 AND PD<1999
L4	3	DUP REM L3 (2 DUPLICATES REMOVED)
L5	12373	S L1 (P) ANGIOGENESIS
L6	5	S L5 AND PD<1999
L7	3	DUP REM L6 (2 DUPLICATES REMOVED)
L8	42595	S (BASIC AND FIBROBLAST AND GROWTH AND FACTOR)/AB
L9	4	S L8 AND L1
L10	0	S L9 AND PD<2000
L11	459	S DEMETHOXYCURCUMIN
L12	183	S L11 AND PD<2000
L13	2	S L12 AND ANGIOGENESIS
L14	1	DUP REM L13 (1 DUPLICATE REMOVED)
L15	9371	S (TUBEROUS AND SCLEROSIS)/AB
L16	3461	S L15 AND PD<2000
L17	1	S L16 AND ANGIOGENESIS
L18	4	S L15 AND L11
L19	3	DUP REM L18 (1 DUPLICATE REMOVED)
L20	0	S L19 AND PD<2000
L21	75667	S (TUBEROUS AND SCLEROSIS)
L22	6	S L21 AND L11
L23	0	S L22 AND PD<2000
L24	23	S CURCUMINOID AND ANGIOGENESIS
L25	0	S L24 AND PD<2000
L26	6	S L8 AND L11
L27	4	DUP REM L26 (2 DUPLICATES REMOVED)
L28	82138	S (BASIC AND FIBROBLAST AND GROWTH AND FACTOR)
L29	10	S L28 AND L11
L30	6	DUP REM L29 (4 DUPLICATES REMOVED)

(FILE 'HOME' ENTERED AT 09:40:38 ON 31 OCT 2003)

FILE 'ADISCTI, ADISINSIGHT, ADISNEWS, BIOSIS, BIOTECHNO, CANCERLIT, CAPLUS, CEN, DISSABS, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, DRUGU, EMBAL, EMBASE, ESBIOBASE, IFIPAT, IPA, JICST-EPLUS, KOSMET, LIFESCI, MEDICONF, MEDLINE, NAPRALERT, NLDB, NUTRACEUT, ..' ENTERED AT 09:40:57 ON 31 OCT 2003

L1	13191	S (DEMETHOXYCURCUMIN OR CURCUMINOID OR CURCUMIN)
L2	73	S L1 AND ANTIANGIOGENIC
L3	31	DUP REM L2 (42 DUPLICATES REMOVED)
L4	2	S L3 AND PD<1999
L5	70870	S BASIC (W) FIBROBLAST (W) GROWTH (W) FACTOR
L6	32271	S L5 AND PD<2000
L7	15239	DUP REM L6 (17032 DUPLICATES REMOVED)
L8	2	S L7 AND L1
L9	0	S L7 AND DEMETHOXYCURCUMIN
L10	2	S (ANGIOSTATIC) AND DEMETHOXYCURCUMIN
L11	1	DUP REM L10 (1 DUPLICATE REMOVED)
L12	16	S (ANGIOGENIC) AND DEMETHOXYCURCUMIN
L13	7	DUP REM L12 (9 DUPLICATES REMOVED)
L14	0	S L13 AND PD<2000
L15	9648	S HEMANGIOENDOTHELIOMA
L16	3515	S L15 AND PD<2000
L17	123	S L15 AND L5
L18	77	DUP REM L17 (46 DUPLICATES REMOVED)
L19	13	S L18 AND L16
L20	12	S L19 AND ANGIOGENESIS
L21	113416	S MALIGNANT (W) MELANOMA
L22	2040	S L21 AND (ANGIOGENESIS OR ANTIANGIOGENIC OR ANGIOSTATIC)
L23	1422	DUP REM L22 (618 DUPLICATES REMOVED)
L24	274	S L23 AND PD<2000

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      (FILE 'HOME' ENTERED AT 11:41:43 ON 31 OCT 2003)

      FILE 'ADISCTI, ADISINSIGHT, ADISNEWS, BIOSIS, BIOTECHNO, CANCERLIT,
      CAPLUS, CEN, DISSABS, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, DRUGU,
      EMBAL, EMBASE, ESBIOBASE, IFIPAT, IPA, JICST-EPLUS, KOSMET, LIFESCI,
      MEDICONF, MEDLINE, NAPRALERT, NLDB, NUTRACEUT, ...' ENTERED AT 11:42:06
      ON 31 OCT 2003
L1           808 S KARPOSI AND SARCOMA
L2           436 S L1 AND (ANGIOGENESIS OR ANTIANGIOGENIC OR ANGIOSTATIC)
L3             5 S L2 AND PD<2000
L4         63078 S KAPOSI AND SARCOMA
L5           436 S L2 AND (ANGIOGENESIS OR ANTIANGIOGENIC OR ANGIOSTATIC)
L6             5 S L5 AND PD<2000
L7             0 S L6 NOT L3
L8         27592 S (KAPOSI AND SARCOMA)/AB
L9            49 S (KARPOSI AND SARCOMA)/AB
L10        27626 S L8 OR L9
L11        10674 S L10 AND PD<2000
L12         5053 DUP REM L11 (5621 DUPLICATES REMOVED)
L13          436 S L2 AND (ANGIOGENESIS OR ANTIANGIOGENIC OR ANGIOSTATIC)
L14          187 S L12 AND (ANGIOGENESIS OR ANTIANGIOGENIC OR ANGIOSTATIC)
L15          148 S L14 AND (ANGIOGENESIS OR ANTIANGIOGENIC OR ANGIOSTATIC)/AB
L16          148 S L15 AND (SARCOMA)/AB
L17          148 S L16 AND PD<2000
L18           35 S L17 AND (FIBROBLAST)/AB
L19           25 S L18 AND BASIC/AB
L20            0 S L19 AND (CURCUMIN OR CURCUMINOID OR DEMETHOXYCURCUMIN)/AB
L21            0 S L19 AND (CURCUMIN OR CURCUMINOID OR DEMETHOXYCURCUMIN)

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